medical management, with rest in bed. The local condition of his ulcer will be better, and his general condition will be improved.

I think it is now generally conceded that all patients with chronic, indurated gastric ulcers should be treated surgically. However, gastro-enterostomy alone will cure only a small percentage of them. The radical resections should be reserved for those patients where the indications justify the risk of radical treatment. The last word in gastric surgery has not been said.

It is well to bear in mind the high percentage of cures achieved by well-established methods. By improving our technique, as well as by more carefully selecting our cases, we can still further increase this percentage.

## AREA CHANGES IN HEARTS SHOWING DECOMPENSATION AND LOWERED CARDIAC RESERVE, WITH REPORT OF TWENTY CASES

*By* DONALD J. FRICK, M. D., ROBERT H. KENNICOTT, M. D., ROLLA G. KARSHNER, M. D., *Los Angeles*

*Cardiac areas, as computed in the orthodiagram, show a fluctuation during compensation and decompensation.*

*In cases of lowered cardiac reserve, enlarged hearts are seen to decrease in size with clinical improvement.*

*In a small number of cases, subsequent enlargement of cardiac area accompanying clinical improvement and resumption of exercise would point to cardiac hypertrophy.*

*Correlation of cardiac areas and clinical findings give information valuable in determining treatment and indicating prognosis.*

DISCUSSION *by F. F. Gundrum, Sacramento; William J. Kerr, San Francisco; A. W. Hewlett, San Francisco; Harry Spiro, San Francisco; F. R. Nuzum, Santa Barbara.*

THE diversity of opinion regarding the variation in heart size, which accompanies changes in compensation, has largely resulted from statements based on inexact data. We have attempted in this paper to so correlate clinical observations and laboratory findings that a clear vision of the subject may be attained. For some time we have felt convinced that the heart area does alter with changes in cardiac function. With this in view, we have selected twenty cases of enlarged hearts, without reference to their outcome, and demanding only that sufficient study had been made upon them to show what, if any, change had taken place during the course of disease. The minimum time of observation of these cases was two months. The maximum was five years and five months. The average period of observation was one year and five months.

Of these twenty cases, eleven showed clinical signs of decompensation at some time during observation. The remaining nine cases showed evidence of lowered cardiac reserve, but no symptoms of decompensation. We here determine decompensation by the clinical signs of venous stasis. In those cases classified under the heading of lowered cardiac reserve, anginal pain and shortness of breath on exertion were the most frequent symptoms.

The cardiac areas were computed from orthodiagrams after the method of Van Zwaluwenburg. Here the formula for computing the area of an ellipse was utilized; namely, the product of the long diameter drawn through the center of the figure and the short diameter erected perpendicular thereto at the widest portion multiplied by the factor 0.7854. Van Zwaluwenburg demonstrated the accuracy of this method by a comparison of areas determined by this method and by planimeter readings. More recently, Karshner and Kennicott, after a study of one hundred normal and abnormal cases, showed an average variation of 2.6 per cent between areas determined by planimeter readings and those figured on a basis of the formula of Van Zwaluwenburg.

Using this very accurate method of measurement of heart areas as a standard, we have demonstrated in seven of the eleven cases of cardiac decompensation a definite decrease in area accompanying reestablishment of compensation. Two of these eleven cases showed progressive increase in size as the hearts gradually failed. One heart showed no change with clinical improvement, and one increased in area.

Of the nine cases of lowered cardiac reserve with cardiac enlargement, five showed decreased heart areas upon clinical improvement; three remained unchanged; while the one remaining heart increased in area as cardiac reserve improved.

In fourteen of the twenty cases, a change in heart area was demonstrated, while four remained unchanged and two increased in size with clinical improvement.

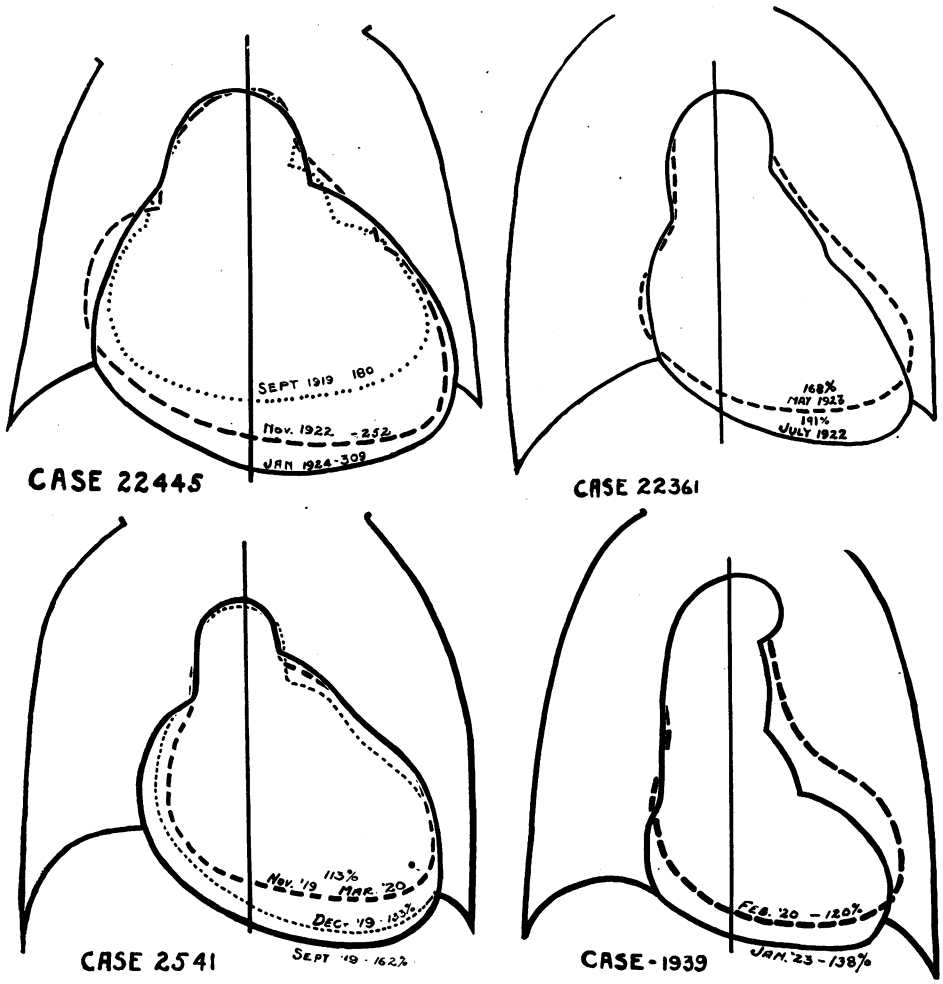
In the entire series, four cases at some time showed increase in heart areas accompanying clinical improvement. These probably demonstrate the result of muscular hypertrophy. The improved tone of the heart is readily observed during fluoroscopic study. The complete orthodiagrametric report in such cases throws more light on the reason for the increased size than a simple statement of area in percentage would indicate. Even among those cases of cardiac decompensation where a definite decrease in cardiac size has followed compensation, there are a few cases which have shown a progressive increase in size upon a resumption of activity following treatment with rest. The area in these cases never reaches the height reached during the period of decompensation, and the tone of the muscle observed during examination shows progressive improvement.

The accompanying chart shows in detail, under the date of examination, the diagnosis, etiology, electrocardiographic and polygraphic diagnosis, orthodiagrametric findings, and clinical observations in the twenty cases studied.

The diameters M. R. and M. L. and total diameters are included, since they are the ones commonly employed by the clinician in his study of the heart. These measurements give information of comparative value, but fall far short of measurements of cardiac area as a means of studying changes in the size of the heart.

The first diagram represents the orthodiagramatic findings of a normal heart. In computing cardiac areas, the long diameter A. B. (in centimeters) is multiplied by the short diameter C. D. (in centimeters), and the product thus obtained is divided by the normal obtained in a similar manner based on body weight. The perpendicular line represents the anatomical center of the body. The M. R. and M. L. are raised perpendicular to this central line at the point of greatest cardiac width to the right and left.

The diagrams were prepared by making a composite picture of the actual orthodiagrams. They demonstrate the changes in the cardiac silhouette




during the course of disease. The areas computed as described above are recorded at the base of the outline in each case.

**Case No. 2541**—This patient was a housewife of 50. She reported September 29, 1919, complaining of dyspnoea and orthopnoea of two weeks' duration. With the exception of repeated attacks of tonsillitis and typhoid fever at the age of 34, the patient had been in good health until February of 1918, when she noted a sudden irregularity of her heart. Physical examination revealed a mitral insufficiency and stenosis with an auricular fibrillation. Her orthodiagram at this time (September, 1919) showed an area of 162 per cent, and is represented by the outer solid line in the diagram. By November 17, 1919, the patient had improved; her pulse, still irregular, was slowed and no longer showed a deficit. Her total transverse diameter was reduced 2.5 cm., and her cardiac area reduced 49 per cent (represented by the heavy, broken line). On December 23, 1919, the patient again reported, showing signs of decompensation; her cardiac area had increased to 133 per cent, shown by the small, broken line on the slide. By March 29, 1920, the patient was greatly improved, her heart had receded to its previous area of 116 per cent, which is virtually that of November of 1919, and shown as the heavy, broken line.

**Case No. 1939** is one of a physician of 58. The diagnosis was luetic aortitis, cardiac dilatation and hypertrophy, chronic nephritis with hypertension, and chronic myocarditis. He presented a right bundle branch block. He had repeated attacks of pulmonary oedema, finally dying in one in January, 1924. He was first seen in March, 1917. He gave a history of having had two attacks of acute oedema of the lungs in January and March. He was complaining of dyspnoea on exertion. He had a blood pressure of 210 systolic and 165 diastolic. His blood Wassermann was three plus. After being on anti-luetic treatment, he reported much improved on June 26, 1917. His first orthodiagram made in February, 1920, gave an area of 120 per cent (represented by the inner broken line), and his fluoroscopic examination revealed a uniformly broad aortic shadow 6 cm. wide. He was feeling well, and working at this time. His antiluetic treatment was continued. January, 1922, patient reported at office. He was definitely failing in health. His electrocardiograms showed ventricular extrasystole with right bundle branch block. His cardiac area had increased to 138 per cent (represented by the outer solid line), an increase of 18 per cent. In December, 1922, he had a severe attack of hemorrhagic oedema of the lungs. The patient grew progressively worse and died in January, 1924, with acute hemorrhagic oedema of the lungs.

**Case No. 22,445**—A nurse, aged 51, came to the office in August of 1919, complaining of "heart trouble." Her previous illnesses were scarlet fever, tonsillitis, typhoid fever, malaria, and rheumatism. A diagnosis was made of mitral and aortic insufficiency, chronic myocarditis, and auricular fibrillation. In September of that year her heart was compensated. Her cardiac area was 180 per cent (inner dotted line). She was on duty as a nurse. In September, 1922, she was suffering from decompensation, with ascites, oedema of legs, cyanosis, and enlarged liver. Her cardiac area was 252 per cent of the normal (the broad, broken line). The patient was improved by February, 1923, but never regained her compensation. Hers was a history of steady decline. When last seen, in January of 1924, she was able to get about a little, but was greatly restricted as compared with her former activities. Her cardiac area was 309 per cent, and is shown in the diagram by the solid line.

**Case No. 22,361**—A man of 42, first seen in July, 1919, complaining of cough, dyspnoea (nocturnal, and on exertion), and precordial pain. He had a decompensated heart, with mitral regurgitation and chronic myocarditis. His cardiac area was 191 per cent. In October, 1922, after

| No. | Age | Diagnosis | Etiology | Dates | E. K. G. and Polygraphs | Heart Measurements M. R. | M. L. | Total | Area | Condition | Remarks |
|---|---|---|---|---|---|---|---|---|---|---|---|
| 2541 | 50 | Mitral disease. Cerebral embolism. | Tonsillitis. Typhoid fever. | Sept. 29, 1919 | | 5.6 | 9.9 | 15.5 | 162% | Decompensation. | Dyspnoea. Orthopnea. Radial pulse 80; apex 96; deficit 16. |
| | | | | Nov. 17, 1919 | | 3.8 | 9.9 | 13.7 | 116% | Improved. | Pulse 72, no deficit. Patient improved. |
| | | | | Dec. 23, 1919 | | 4.6 | 10.1 | 14.7 | 133% | Signs of decompensation. | Distress after eating. |
| | | | | Mar. 29, 1920 | | 5.0 | 9.0 | 14.0 | 116% | Improved. | |
| | | | | May 14, 1920 | | 5.0 | 9.0 | 14.0 | 116% | Improved. | Patient able to shop. |
| | | | | Nov. 23, 1920 | | 5.0 | 9.0 | 14.0 | 116% | Improved. | Free from cardiac symptoms. |
| | | | | May 25, 1922 | | ..... | ..... | ..... | ..... | Death. | Cerebral embolism. |
| 1939 | 58 | Luetic aortitis. Cardiac dilatation and hypertrophy. Oedema lungs. Hypertension. Right branch block. Chronic nephritis. Myocarditis, chronic. | Syphilis. | Feb. 27, 1920 | | 4.8 | 8.8 | 13.6 | 120% | ..... | Wassermann ++++. |
| | | | | Mar. 21, 1920 | | ..... | ..... | ..... | ..... | ..... | Reports 3 attacks oedema of lungs. |
| | | | | Jan. 19, 1922 | Left ventricular extra systole. Right branch block. Notched Q R S in Leads I, II and III. | 4.1 | 9.4 | 13.5 | 138% | Failing. | Wassermann ++++. Mercury and K. I. |
| | | | | Dec. 13, 1922 | | ..... | ..... | ..... | ..... | ..... | Severe attack hemorrhagic oedema of lungs. |
| | | | | Jan. —, 1924 | | ..... | ..... | ..... | ..... | Death. | Pulmonary oedema. |
| 21362 | 42 | Myocarditis, chronic. Heart block. | Tonsillitis. | July 20, 1921 | Heart block 2 to 1. | 4.2 | 11.5 | 15.7 | 167% | Decompensated. | Fatigue and pain in chest after exercise. |
| | | | | Sept. 6, 1921 | Heart block 2 to 1. | 5.2 | 9.5 | 14.7 | 150% | Improved. | Walking 15 blocks and climbing stairs without distress. On salvarsan. |
| | | | | Oct. 26, 1921 | | ..... | ..... | 13.6 | 114% | Improved. | Course of mercury succinamide. |
| | | | | Dec. 16, 1921 | | ..... | ..... | 15.7 | 119% | Condition good. | |
| 1285 | 62 | Tonsillitis, chronic. Mitral stenosis. Auricular fibrillation. | Influenza. Tonsillitis, chronic. | Sept. 5, 1918 | Auricular fibrillation. Inverted T in Lead II. | | | | | | |
| | | | | Nov. 25, 1919 | | 3.7 | 7.4 | 11.1 | 94% | ..... | Precordial pain with shortness of breath on exertion. |
| | | | | Nov. 20, 1923 | Auricular fibrillation (fine). Slight right axis deviation. | | | | | | |
| | | | | Feb. 19, 1924 | | 4.5 | 8.5 | 13.0 | 120% | Exceptionally well. | Back at work. |
| 22267 | 59 | Angina pectoris. Arteriosclerosis. Abscess alveolaris. Pericarditis, acute. Pleurisy. | Arthritis, 8-10. Alveolaris abscess. | June 27, 1922 | | 5.1 | 11.7 | 16.8 | 151% | Acutely ill. | Pleurisy with pericarditis with acute anginal attack. Four abscessed teeth. |
| | | | | Aug. 26, 1922 | | ..... | ..... | ..... | ..... | ..... | Abscessed teeth removed. |
| | | | | Nov. 10, 1922 | | 4.6 | 10.4 | 15.0 | 135% | Much improved. | |
| | | | | Dec. 1, 1922 | Notching Q R S L. II. Left ventricular preponderance. Inverted T Lead I T opposite to main deflection in Leads I and III. | | | | | | |
| | | | | Feb. 8, 1923 | | 5.0 | 11.2 | 16.2 | 141% | Improved. | |
| | | | | Dec. 16, 1923 | | ..... | ..... | ..... | ..... | Improved. | Walking one mile a day. Enjoying his work. |
| 2801 | 31 | Mitral stenosis et insufficiency. Alveolar abscess. Paroxysmal tachycardia. | Influenza, 30. Alveolar abscess. | May 13, 1920 | | 4.8 | 10.5 | 15.3 | 194% | Compensated. | No symptoms. Was told she had heart trouble. |
| | | | | Jan. 27, 1921 | Polygraph-paroxysmal tachycardia, rate 190, regular. | 4.8 | 10.5 | 15.3 | 244% | Decompensated. | Dullness in both flanks. Attack of tachycardia. |
| | | | | Dec. 23, 1921 | | 4.8 | 9.0 | 13.3 | 187% | Improved. | Walking short distances. |
| 819 S | 50 | Endocarditis, chronic. Auricular fibrillation. | Arthritis, age 15. | Jan. 2, 1920 | | ..... | ..... | ..... | ..... | Decompensation. | Orthopnea. Dyspnoea. Rales in both bases. |
| | | | | Feb. 4, 1920 | | 4.6 | 9.8 | 14.4 | 172% | Improved. | |

| No. | Age | Diagnosis | Etiology | Dates | E. K. G. and Polygraphs | Heart Measurements M. R. | M. L. | Total | Area | Condition | Remarks |
|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | Feb. 18, 1920 | | 4.2 | 9.3 | 13.5 | 138% | Continued improvement. | Lungs clear. |
| | | | | Oct. 6, 1920 | | 4.5 | 9.9 | 14.4 | 147% | Moderate decompensation. | Slight nocturnal dyspnoea. Blood pressure gradually declining. 96/80. |
| | | | | Oct. 19, 1920 | | 4.6 | 9.1 | 13.7 | 135% | Improved. | |
| | | | | Feb. 9, 1921 | | 4.6 | 10.3 | 14.9 | 183% | Decompensation. | Acute bronchitis in January. Heart absolutely irregular. Rales in both bases. |
| | | | | May 25, 1921 | | 5.5 | 10.1 | 15.6 | 168% | Improved. | |
| | | | | Sept. 3, 1921 | | ..... | ..... | ..... | ..... | Decompensated. | Failing rapidly. |
| | | | | Sept. 27, 1921 | | ..... | ..... | ..... | ..... | Death. | |
| 22445 | 51 | Mitral and aortic insufficiency. Myocarditis. Auricular fibrillation. | Tonsillitis. Rheumatism. | Sept. —, 1919 | | 7.5 | 10.0 | 17.5 | 180% | Compensatory. | Working as nurse. |
| | | | | Sept. —, 1922 | | 8.8 | 10.6 | 19.4 | 252% | Decompensated. | Ascites. Oedema of lungs. Dyspnoea, cyanosis and enlarged liver. |
| | | | | Feb. 9, 1923 | Auricular fibrillation. Right ventricular preponderance. Rate 69. Inverted T in Leads III. Notching of Q R S in all Leads. | ..... | ..... | ..... | ..... | Improved. | Liver enlarged. Fluid in flanks. |
| | | | | Dec. 15, 1823 | Auricular fibrillation. Right ventricular preponderance. Rate 54. Notching Q R S all Leads. Right ventricular extra systole. Diminutive deflection. | ..... | ..... | ..... | ..... | Gradually failing. | In hospital with acute respiratory infection. |
| | | | | Jan. 23, 1924 | | 8.3 | 11.2 | 19.5 | 309% | Failing. | Doing very little. |
| 22153 | 70 | Myocarditis. Ventricular extra systole. Paroxysmal auricular fibrillation. | Influenza. Tonsillitis. Arthritis. Typhoid, 35. | Mar. 25, 1922 | Polygraph—rate 103. Ventricular extra systole with alternation following. | 2.8 | 10.0 | 12.8 | 160% | Mild decompensation. | Palpitation. Dyspnoea on exertion, and weakness. |
| | | | | Dec. 8, 1922 | E. K. G. Ventricular extra systole right and left. Inverted T in Lead III. | | | | | | |
| | | | | Jan. 4, 1923 | | 2.8 | 10.0 | 12.8 | 160% | Improved. | |
| 2919 | 45 | Mitral stenosis. Regurgitation. Tonsillitis, chronic. | Tonsillitis. | Aug. 26, 1920 | | 4.0 | 10.2 | 14.2 | 194% | ..... | Shortness of breath. Precordial pain and palpitation on exertion. |
| | | | | Sept. 20, 1920 | | ..... | ..... | ..... | ..... | ..... | Tonsillectomy. |
| | | | | June 20, 1921 | | 4.7 | 10.5 | 15.2 | 194% | No improvement. | Marked shortness of breath on exertion. |
| | | | | May —, 1923 | | ..... | ..... | ..... | ..... | ..... | Cardiac—death. |
| 3037 | 34 | Myocarditis. Auricular extra systole. | Typhoid fever, age 24. Hyperthyroidism, age 18. | Dec. 23, 1920 | Polygraph—alternate auricular extra systole. | 3.0 | 7.5 | 10.5 | 124% | Moderate decompensation. | Rapid irregular heart. Precordial pain. Slight dyspnoea. |
| | | | | Aug. 15, 1921 | | 4.5 | 7.0 | 11.5 | 144% | Improved. | Heart slower. Better compensation. Walking 1½ miles. |
| | | | | Mar. —, 1924 | | ..... | ..... | ..... | ..... | ..... | General health best for 10 years. |
| 2667 | 54 | Mitral insufficiency. Auricular fibrillation. | Tonsillitis, chronic. Broncho-pneumonia, 54. Typhoid, 14. Myositis, chr. | Jan. 21, 1920 | | 4.2 | 11.0 | 15.2 | 130% | Compensatory. | General debility following pneumonia. |
| | | | | Nov. 10, 1921 | | 4.0 | 10.2 | 14.2 | 113% | Improved. | |
| 2446 | 52 | Toxic hyperplastic goitre. Myocarditis. Nephritis, chronic. | Chorea, 13. Tonsillitis. Goitre, 40. | Sept. 23, 1919 | | ..... | ..... | ..... | ..... | ..... | Partial thyroidectomy. |
| | | | | Feb. 10, 1920 | | 2.0 | 9.4 | 11.4 | 125% | Improved. | Doing a little work. Heart rapid. |

| No. | Age | Diagnosis | Etiology | Dates | E. K. G. and Polygraphs | Heart Measurements M. R. | M. L. | Total | Area | Condition | Remarks |
|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | Mar. 4, 1921 | | 2.0 | 9.4 | 11.4 | 125% | No improvement. | No loss of compensation. Tremor. Sweating. Diarrhoea. Heart 142 per minute at times. |
| 21080 | 48 | Mitral stenosis et insufficiency. Auricular fibrillation. | Tonsillitis. Alveolar abscess. Pyorrhoea. Colecystitis and colelithiasis. | Feb. 8, 1921 | | 6.5 | 12.3 | 18.8 | 286% | Decompensated. | Dyspnoea on exertion. Orthopnea. Liver 10 cm. below costal border. Oedema of legs to thighs. |
| | | | | Apr. 15, 1921 | | 5.7 | 11.7 | 17.4 | 210% | Improved. | Slight pretibial oedema. Liver smaller, still palpable. |
| 21085 | 36 | Chronic nephritis with N and NaCl retention. Myocarditis, chronic. Hypertension. | Influenza, 33. Antrum, 24. Nephritis, 36. | Sept. 2, 1921 | | 4.7 | 11.1 | 15.8 | 184% | Decompensated. | Precordial distress on climbing hills. Slight oedema. Dyspnoea. |
| | | | | Nov. 4, 1921 | | 4.1 | 10.7 | 14.8 | 170% | Improved. | |
| | | | | ——— , 1922 | | ..... | ..... | ..... | ..... | ..... | Death—nephritis. |
| 22103 | 42 | Myocarditis, chronic. Mitral insufficiency. Partial heart block. | Influenza, 39. Otitis media, 42. Tonsillitis, chr. Infantile paralysis, 3. | Feb. 27, 1922 | | 2.7 | 10.2 | 12.9 | 151% | Weak following otitis media. | Slow, irregular pulse. Hypertension. No cardiac symptoms. |
| | | | | May 5, 1922 | Delayed conduction time. Partial heart block. Notching Q R S Lead III. | 2.2 | 10.2 | 12.4 | 139% | Improved. | Patient free from symptoms. |
| 23211 | 55 | Myocarditis, chronic. Angina pectoris. | Influenza, 55. | Feb. 19, 1923 | | 3.2 | 10.5 | 13.7 | 118% | Compensated. | Tracing made in France. |
| | | | | Apr. 26, 1923 | | 2.6 | 11.2 | 13.8 | 118% | Improved. | Compensation good. Tracings made here on return from abroad. |
| 21151 | 72 | Nephritis, chronic. Myocarditis, chronic. General arteriosclerosis. Herpes zoster. | Tbc. at 12. | Mar. 16, 1921 | | 5.4 | 13.0 | 18.4 | 201% | Decompensated. | Ten days previous had sudden brief loss of consciousness, preceded by dizziness and dyspnoea. Liver enlarged. |
| | | | | Apr. 26, 1921 | | 4.9 | 11.9 | 16.8 | 175% | Improved. | Urea dropped from 26 to 20. |
| | | | | June 30, 1921 | | 5.9 | 12.3 | 18.2 | 194% | Good. | Cardiac reserve good. Liver palpable. |
| 22410 S | 39 | Hyperthyroidism. Myocarditis. Tonsillitis, chronic. | Tonsillitis. Hyperthyroidism. | Aug. 22, 1922 | | 3.9 | 9.2 | 13.1 | 127% | ..... | Basal metabolism +44%. Rapid pulse 100-120. Loss of weight. Shortness of breath. |
| | | | | Sept. 26, 1922 | | ..... | ..... | ..... | ..... | ..... | Tonsillectomy. |
| | | | | Oct. 22, 1922 | | ..... | ..... | ..... | ..... | ..... | Thyroidectomy. Basal metabolism +11%. |
| | | | | Oct. 24, 1922 | | 3.7 | 9.3 | 13.0 | 121% | Improved. | |
| | | | | Nov. 21, 1922 | | ..... | ..... | ..... | ..... | ..... | Basal metabolism +.5%. 4/5 thyroid removed. |
| | | | | Feb. 16, 1923 | | 4.5 | 8.7 | 13.2 | 111% | Much improved. | |
| 22361 | 42 | Myocarditis. Mitral regurgitation. | Arthritis, 11. Influenza, 39. | July 17, 1919 | | 4.2 | 11.0 | 15.2 | 191% | ..... | Cough, dyspnoea (nocturnal and on exertion). Precordial pain. |
| | | | | Oct. 9, 1922 | | 4.6 | 11.0 | 15.6 | 165% | Improved. | Free from dyspnoea. No precordial pain. |
| | | | | Feb. 2, 1923 | Left ventricular extra systole. Notching of Q R S in Lead III. | ..... | ..... | ..... | ..... | Improved. | Limited activity. Four hours' work in office. |
| | | | | May 16, 1923 | | 4.6 | 11.0 | 15.6 | 168% | Improved. | Shape of heart improved. |
| | | | | Feb. 1, 1924 | | 5.0 | 11.3 | 16.3 | 185% | Improved. | Free from signs of decompensation. Slight dyspnoea on exertion. 12 hours' activity. 2 hours' rest in bed. |

treatment, the patient was free from symptoms and presented a cardiac area of 165 per cent. Improvement continued and the patient was allowed limited activity and four hours' work at his office daily. His electrocardiograms were negative but for ventricular extrasystoles. In May, 1923, his heart was slightly larger (168 per cent), but of definitely better tone. This heart outline is represented by the broken line in the illustration. When last seen in February, 1924, the patient was free from signs of decompensation. He had slight dyspnoea on exertion. He was active about twelve hours a day, with two hours' rest in the afternoon. His heart was definitely larger than at the previous examination (185 per cent), but his muscle tone was good.

## CONCLUSIONS

1. That cardiac areas, as computed in the orthodiagram, show a fluctuation during compensation and decompensation.
2. That in cases of lowered cardiac reserve enlarged hearts are seen to decrease in size with clinical improvement.
3. That in a small number of cases subsequent enlargement of cardiac area accompanying clinical improvement and resumption of exercise would point to cardiac hypertrophy.
4. That correlation of cardiac areas and clinical findings give information valuable in determining treatment and indicating prognosis.

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## DISCUSSION

F. F. GUNDRUM, M. D. (Capital National Bank Building, Sacramento)—I have been very pleased with this paper. The publication of such accurate, painstaking, and continuous study of heart size will throw a very needed light upon our hitherto somewhat hazy concepts of the relation of cardiac dimensions to cardiac ability. We have long been taught that heart weakness was associated with dilatation, and I believe it was Osler who said that endurance was to be measured by the ability of the heart to resist this constant tendency to dilate. With more accurate study, as appears in Dr. Frick's paper and elsewhere, it begins to seem evident that this idea will have to be somewhat revised, and endurance may prove to be, in large measure, a function of blood-sugar. Although we are all daily seeing examples of the reduction in the width of cardiac dullness which accompanies the recovery from heart failure, increase in the ability of the heart to do work is not always associated with a decrease in heart size. It is hardly necessary to use such elaborate technique to make a clinical diagnosis of heart failure, and to get a quite accurate picture of the general outline of the heart. However, this sort of research will prove invaluable in giving us the exact relations between those very small differences in heart size and shape (if there are any) which are associated with the minor degrees of heart insufficiency, and perhaps foretell a cardiac breakdown.

WILLIAM J. KERR, M. D. (University Hospital, San Francisco)—The report by Drs. Frick, Kennicott, and Karshner is of interest to physicians in general and to cardiologists in particular, because it emphasizes the value of accurate methods of determining cardiac measurements. We cannot rely on our present methods of physical examination for the accurate sizes of hearts, particularly in those cases where emphysema is a complication or in individuals who have an unusual amount of subcutaneous fat. Here the orthodiagraphic method comes into use.

The authors have shown the variation in cardiac area over a long period of time in their series of cases, and their charts definitely show that the variations are usually associated with changes in the cardiovascular status of the patient. The value of this method in treatment and in prognosis seems to me to be very great and should be more generally employed. One should be extremely careful, I think, to have the orthodiagrams taken of the patient in the same position each time so that changes in shape and position of the heart by changes and position in the patient will be obviated or minimized.

I feel that the study of the heart with the area of the orthodiagraphic shadow and the orthodiagrapic plate before us for changes in contour give a very good idea of the conditions present, and is much more satisfactory than either method alone. These methods do not replace the other methods of physical diagnosis and examination of the patient, but are useful aids in the proper treatment and prognosis of the case.

A. W. HEWLETT, M. D. (Stanford University Medical School, San Francisco)—This paper demonstrates the value of accurately measuring the size of the heart shadow by means of the x-ray. The difficulty encountered in making accurate measurements of the heart shadows in different patients arises from the considerable variations encountered in normal persons. On account of these variations, the heart shadow must be considerably larger than the average before we are certain that it exceeds the upper limit of normal. In the present paper, comparative observations were made on the same individuals at different times, so that small changes in area are significant. These comparative measurements show clearly that, in certain patients, the heart shadow lessens as compensation improves. From my own experience with chest plates of cardiac patients, I can confirm the observation that certain patients show a decrease in the heart shadow coincident with clinical improvement, while others show little or no change. The authors report four cases in which, at some time, the heart shadow became larger during clinical improvement. They attribute this to cardiac hypertrophy. It appears to me improbable that a marked change, such as that observed in Case 1285, could be due entirely to thickening of the heart muscle. Increase of the heart shadow, especially in diastole, may be caused by a slower heart rate. It is also probable that, in some instances, hypertrophy goes hand in hand with a moderate dilatation even when compensation is improving.

HARRY SPIRO, M. D. (Flood Building, San Francisco)—The orthodiagraphic method of cardiac examination has had a hard path to travel. Popular prejudice decided that the method was too tedious, required extraordinary skill and high-priced and complicated apparatus; therefore, it has not been more in demand. But no sooner does an individual decide upon a careful and thorough study of cardiac conditions than he realizes that, for dependable work, orthodiagrams are an indispensable supplement to the radiograms.

Today the orthodiagraphic apparatus is very simple and does not demand more than ordinary skill to operate and interpret. Its value is tremendous, particularly as an aid in diagnosing borderline cases of cardiac defects. The authors have shown further that it may be a factor in preventing a cardiac breakdown because one can recognize changes in size and shape of the heart which often precede cardinal symptoms of severe cardiac distress.

While in general I do not believe that cardiac shape is dependable in judging cardiac muscle tone, still there are cardiac types which are reliably associated with certain cardiac defects. For instance, if an orthodiagraphic study has shown a certain type heart, and after a period of time investigation shows a distinct deviation from the type previously found, one could reasonably assume that the heart was not standing up to its work properly, and the treatment should be changed accordingly.

We have always believed that progressive increase in cardiac size spelled approaching trouble. On the contrary, the authors have called attention to the possibility that if a decompensated heart improves and decreases in size, and subsequently as the improvement continues, the heart is shown by careful measurements to be increased in size; then this latter increase in size is due to healthy changes and need not be feared. The proof of the above is definitely a step forward, and shows with what thoughtful judgment the authors performed their work and illustrates forcibly the great value of orthodiagraphic study.

Drs. Frick, Kennicott, and Karshner are to be congratulated in their work of blasting another stone from the path of orthodiagraphy and scoring another point for the long neglected, but faithful, hard-working heart.

FRANKLIN R. NUZUM, M. D. (Cottage Hospital, Santa Barbara)—This paper demonstrates in a very convincing manner the aid that is available by the use of accurate methods of determining the size and shape of the heart. The point has been established that the size and shape of the heart varies in the period of time that elapses between broken compensation and compensation, and that a knowl-

edge of this change is of value in diagnosis, prognosis, and treatment.

Quite aside from the object of this paper, the change of the size of the heart as a result of disease, are some observations made recently in Boston upon the change in the size of the heart in trained athletes following severe exertion. The same methods of determining heart measurements were used, and the information gained is very interesting. Heart measurements were made of these athletes before, immediately after and some days after the completion of a 25-mile marathon race. This race, being an annual event, attracts the best distance runners in the country and men who are thoroughly trained for the event. In brief, the width of these hearts decreased an average of 2 cm., as shown by measurements at the conclusion of the race. Within two to five days these hearts had again returned to the size as demonstrated before the race. This is not in harmony with what many believed would happen as the result of a severe strain such as this race must be. It demonstrates again the value of accurate methods in establishing facts.

**The Clinical Teacher and the Medical Curriculum** —Harvey Cushing, Boston (Journal A. M. A.), says there are fashions in teaching, like fashions in other things, and one must conform or be regarded as out of date, even though, after all, we may reach our destination whether we ride side-saddle or ride astride. Just now, for example, in our medical schools the "didactic lecture" is taboo. Who first put the taboo on lecturing was probably someone in authority incapable of holding the attention of a group of students by this method. Individuality is now submerged: our teaching, must—to use a greatly abused word—be standardized, as though our schools were factories. The personal influence of the teacher has largely become swamped, and we try vainly to atone for this by juggling with the curriculum, forgetful that no two instructors in any two schools can possibly reach students with precisely the same methods; and that no two students get their inspiration, such as it is, in the same way out of their particular school or its individual instructors. I presume the Harvard Medical School is no different from most medical schools, in that no faculty member is quite satisfied with the existing curriculum and, as a result, about every three years some one protests with sufficient energy to force on his reluctant colleagues some radical changes. Students can be well grounded through the medium of any course. In any old and established school, the curriculum inevitably becomes hidebound. Meanwhile, we have become fairly well accustomed to the view that subjects beginning with the study of morphology and ending with the clinical specialties must be taught in a given sequence. So far as the curriculum is concerned, our discussions in faculty meeting are given over largely to the struggle for elbow-room between established courses, of which there are too many. We have just been going through one of our triennial turn-overs at the Harvard Medical School, in the endeavor to find out what is wrong with the student and with our method of teaching. This time, pressure has been brought to bear by certain members of the faculty of a philosophical turn of mind, who have discovered that the trouble with the undergraduate is that he has no time for intellectual cogitation. Consequently, at the risk of not meeting our obligations to state board requirements, we have materially cut down our hours of instruction so that the students have their freedom Tuesday afternoon and Thursday afternoon and all day Saturday and Sunday. We have as yet made no statistical study of the amount of rumination they do in these free hours; nor do I think such a study will ever be made, because by the time there are sufficient data to rely on we shall probably have gone back to the old system, or new courses will have crept in to fill up these free afternoons.

# EDITORIALS

## RULES AND FEE SCHEDULE OF DISTRICT OF COLUMBIA MEDICAL ASSOCIATION PLEASE ORGANIZED LABOR

The reaction produced by the new published rules and fee schedule of the District of Columbia Medical Association still reverberates through the daily and periodic press of the country. Many editors constantly criticize the action, while others, and particularly that part of the press identified with organized labor, are pleased at the action taken. The majority of commentators profess to see in the action a "tightening up" of physicians' organizations and the introduction of "business ethics" into their methods and conduct. Some editors castigate the doctors for introducing the "lockout" and a scale of wages and working conditions similar to those of labor unions. Editors of papers sympathetic to labor unions welcome these innovations and predict affiliation of medical associations with the American Federation of Labor as the logical end to this movement.

*Fee Schedule*—The fee schedule is too long to reproduce. Fees vary from $3 to $5000 and upwards. The rules relating to fees are interesting. They are:

1. The following fees shall be charged for professional services, subject, however, to the several rules which are appended: . . .

2. The foregoing table contains the standard fees which *shall be demanded;* they shall be *increased* according to the judgment of the practitioner concerned, in all cases of extraordinary detention or attendance; also in proportion to the importance of the case, of the responsibility attached to it, and to the service rendered when these are extraordinary. They shall be *diminished* at the discretion of the physician when he believes that the patient cannot afford to pay the regular fees, and yet is able to make some compensation; but diminishing the fees except for motives of charity and benevolence is a violation of this regulation.

3. Medical officers connected with the staffs of the hospitals and dispensaries in the District of Columbia shall charge the usual fees for medical services rendered to persons who seek gratuitous services when they are able to pay.

4. Clergymen are not entitled to gratuitous services except when they are in indigent circumstances. Graduates of medicine are not entitled to gratuitous services unless they devote their entire time to the practice of medicine or by reason of age or infirmity have retired from the regular practice of medicine, or unless such graduates of medicine are in indigent circumstances.

5. It is not designed by these regulations to prevent gratuitous services to those who are incapable of making remuneration without distressing themselves or their families.

6. When a physician engaged to attend a case of obstetrics is absent and a second attends the patient, the latter may charge the full fee, but shall relinquish the patient to the first on his return; and in no case shall the second continue to attend except to render indispensable service during the continued absence or disability of the first.

7. When one or more physicians are called in consultation the attending and consulting physician or physicians shall charge at least the ordinary fee for delivery or other services; but when the latter are not detained in attendance they shall only charge the usual fee for consultation.

8. It is recommended that the members of this society